Postpartum Preeclampsia Complicated by Acute Pulmonary Edema

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Peripartum acute pulmonary edema can occur in patients with preeclampsia-eclampsia. Although the pulmonary edema frequently develops in the postpartum setting, the preeclampsia-eclampsia typically occurs antepartum. Postpartum preeclampsia also can occur with acute pulmonary edema following a relatively normal gestation and delivery.

Introduction

Peripartum acute pulmonary edema is a rare but well-described complication in patients with preeclampsia-eclampsia and is associated with a significant increase in maternal and perinatal morbidity and mortality.[1,2] Although the acute pulmonary edema usually occurs in the early postpartum setting, the preeclampsia-eclampsia typically is present antepartum. This report describes a case of postpartum acute pulmonary edema in a patient with postpartum preeclampsia following a relatively normal gestation and delivery.

Case History

A 27-year-old mother came to the emergency department 3 days postpartum with features of preeclampsia and acute pulmonary edema. She initially developed peripheral edema 24 hours postpartum, followed by progressive respiratory insufficiency. When she entered the emergency department, she was in moderate respiratory distress with mild perioral cyanosis. Her examination revealed an initial blood pressure of 178/110, jugular venous distension, pulmonary rales, and 4+ pitting peripheral edema. The initial chest x-ray revealed diffuse bilateral interstitial infiltrates consistent with pulmonary edema and a normal cardiac silhouette. Her ECG showed sinus tachycardia and the initial room air blood gas was pH 7.50, pO_2 54, pCO_2 37, SaO_2 91%. Further laboratory studies included uric acid elevation (9.6 mg/dl), normal BUN and creatinine, no proteinuria on urinalysis, elevated CPK (350 IU/L) with normal MB fraction, and low serum albumin (2.2 g/dl). Her hematocrit was low (29.3%), but not significantly changed from previous studies. Emergent echocardiogram revealed normal left ventricular size and systolic function with moderate mitral insufficiency, normal left atrial size, and no evidence of left ventricular hypertrophy.

The patient's prenatal course was complicated by preterm labor at 27 weeks of gestation, which required hospitalization and therapy with intravenous magnesium sulphate and subcutaneous terbutaline therapy. She was maintained as an outpatient on oral terbutaline until 37 weeks, when tocolytics were stopped. Two and a half weeks later she delivered a full-term baby without complications during labor and delivery. Review of the patient's labor, delivery, and early postpartum course revealed no excessive intravenous fluid administration or blood product transfusions. Her previous obstetrical history included habitual abortions for which she was treated with aspirin and progesterone therapy. Other pertinent medical history included stage II Hodgkin's disease 4 years prior to this admission, treated with mediastinal radiation therapy (4000 rads) and chemotherapy with Adriamycin (216 mg total dose), bleomycin, DTIC, and Velban. The patient had no evidence of recurrence of Hodgkin's disease and no prior history of hypertension, diabetes, renal insufficiency, or cardiopulmonary problems.

The patient was admitted to the coronary care unit and improved dramatically with diuresis and blood pressure control. She also was started on intravenous magnesium sulphate for seizure precautions. She was discharged in excellent condition following 5 days of hospitalization.

Discussion

Possible etiologies of early postpartum pulmonary edema include postpartum cardiomyopathy, pulmonary edema associated with tocolytic therapy, and pulmonary edema in patients with preeclampsia-eclampsia. This patient came to the emergency department 3 days postpartum in acute pulmonary edema and with evidence of preeclampsia (hypertension, elevated uric acid, and generalized edema). In a previous case series describing 37 patients with preeclampsia-eclampsia complicated by pulmonary edema, all patients had severe preeclampsia-eclampsia antepartum.[1] Interestingly, however, most patients in this series developed pulmonary edema in the early postpartum setting (70% within 72 hours after delivery). The incidence of preeclampsia with postpartum onset is not well defined. Simi-

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larly, we are unaware of previous reports of postpartum preeclampsia complicated by acute pulmonary edema, as in our patient.

The possible mechanism(s) of pulmonary edema in patients with preeclampsia is unclear. The patients typically have generalized arterial spasm with elevated systemic vascular resistance.[3] The plasma oncotic pressure, which decreases in normal pregnancy, has an accentuated decline in patients with preeclampsia. This combination of increased left ventricular afterload and decreased plasma oncotic pressure predisposes these patients to pulmonary edema. The postpartum mobilization of extracellular fluids and the frequent administration of excessive intravenous fluids in the peripartum setting may explain why most patients develop pulmonary edema postpartum. Impaired capillary endothelial permeability has also been postulated as a contributing factor in these patients.[4]

The incidence of pulmonary edema associated with tocolytic therapy is as high as 4.4%.[5] Symptoms usually develop dur-

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ing therapy with the agent, although reported cases of pulmonary edema up to 72 hours after discontinuation of therapy have been reported. Our patient developed pulmonary edema almost 3 weeks after discontinuation of oral terbutaline, and it is unlikely that tocolytic therapy played a significant role in her presentation.

Peripartum cardiomyopathy is another possible etiology in patients presenting with postpartum pulmonary edema. These patients typically present within 4 weeks antepartum and 5 months postpartum.[6] Alternatively, the contribution of this patient's previous treatment of Hodgkin's disease with chemotherapy and radiation therapy in her presentation is unclear. Both chemotherapeutic agents and mediastinal radiation therapy can cause myocardial toxicity leading to left ventricular dysfunction.[7-8] Our patient, however, had no evidence of left ventricular dilation or systolic dysfunction on echocardiogram to suggest either peripartum cardiomyopathy or cardiomyopathy secondary to chemotherapy or radiation therapy.

In summary, postpartum acute pulmonary edema is a rare but well-described complication of pregnancy. Multiple possible etiologies including peripartum cardiomyopathy, pulmonary edema associated with tocolytic therapy, and pulmonary edema secondary to preeclampsia-eclampsia need to be considered in these patients. In previous reports of patients with pulmonary edema associated with preeclampsia, all patients had antepartum evidence of preeclampsia. In this case report, we describe a case of early postpartum pulmonary edema in a patient with postpartum preeclampsia following a relatively normal gestation and delivery.

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